84 Nausea and Vomiting

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Definition

Vomiting, or emesis, is the forceful retrograde expulsion of gastric contents from the body. *Nausea* is the unpleasant sensation that precedes vomiting. Nausea frequently is relieved by vomiting and may be accompanied by increased parasympathetic nervous system activity including diaphoresis, salivation, bradycardia, pallor, and decreased respiratory rate. *Retching* ("dry heaves") is the simultaneous contraction of the abdominal muscles and muscles of inspiration that may occur with vomiting.

Vomiting should be differentiated from *regurgitation,* the nonforceful expulsion of gastric contents into the esophagus, and *eructation* (belching), the expulsion of swallowed gastric air. Regurgitation or eructation may be volitional, or result from an incompetent lower esophageal sphincter. Anatomic alterations of the esophagus (by mucosal rings, carcinoma, or diverticula) and disorders of esophageal motility (such as achalasia and diffuse spasm) may simulate vomiting, but the food bolus never reaches the stomach.

Technique

Nausea and vomiting are nonspecific symptoms that occur with a wide variety of metabolic, toxic, inflammatory, and structural disorders (Table 84.1). Determination of cause requires careful assessment of symptoms associated with the episode, in particular, duration, relation to meals, precipitating factors, and contents of the vomitus.

Abrupt onset of nausea or vomiting is usually due to a toxic, infectious, or central nervous system disorder. Acute symptoms lasting for several hours to a few days are found with inflammatory or structural lesions. Chronic symptoms, particularly if they are intermittent, frequently are produced by an obstructive or inflammatory process within the gastrointestinal tract. Chronic vomiting without other systemic symptoms may be self-induced; chronic nausea in the absence of vomiting may be a clue to psychogenic problems.

Vomiting that contains recognizable food and occurs several hours after meals is associated with gastric motility disorders and anatomic obstruction of the gastric pylorus. Vomiting produced by a structural lesion or motility disorders of the small bowel or colon may have no consistent relation to intake.

Projectile vomiting, particularly on arising from sleep, may be due to increased intracranial pressure. Early morning nausea and vomiting is associated with pregnancy. Adverse reactions to medications are among the commonest causes of nausea or vomiting; the symptoms may be acute or chronic, constant or intermittent. Nausea and vomiting can be learned and elicited by previously neutral stimuli, such as the sight of a hospital or physician's office.

Vomitus that contains blood or coffee ground-like material usually indicates bleeding from a source proximal to the ligament of Treitz, but swallowed blood from oropharyngeal or pulmonary lesions may simulate upper gastrointestinal tract bleeding. In the patient who develops vomiting after surgery for peptic ulcer disease, bile-stained vomitus may be a clue to gastric inflammation produced by reflux of alkaline duodenal contents into the stomach. Obstructive lesions of the small bowel may be associated with vomitus containing recognizable food or clear intestinal secretions,

Table 84.1
Conditions Associated with Nausea and Vomiting

Toxic/metabolic
Drugs, chemical toxins (alcohol, narcotics, digitalis glycosides)
Biologic toxins (staphlococcal enterotoxin)
Pregnancy (estrogens)
Uremia
Acidosis/ketosis (diabetic ketoacidosis, alcoholic ketosis)
Electrolyte abnormalities (acute adrenal insufficiency, hypercalcemia)

Infectious
Viral infections
Sepsis
Meningitis/encephalitis

Central nervous system
Increased intracranial pressure (trauma, stroke, meningitis, malignant hypertension, vascular headache)
Idiopathic epilepsy
Vertigo/motion sickness

Gastrointestinal
Anatomic obstruction or motility disorder
- Gastric outlet obstruction (peptic ulcer, carcinoma)
- Small bowel obstruction (adhesion, herniation)
- Colonic obstruction (carcinoma, volvulus)
- Cholecystitis
- Idiopathic pseudoobstruction
- Diabetic gastroenteropathy

Inflammatory
- Gastritis/duodenitis/peptic ulcer disease
- Pancreatitis/hepatitis
- Peritonitis/mesenteric ischemia

Renal
Renal colic
Uremia

Gynecologic
Pregnancy
Premenstrual stress syndrome
Neoplasm

Cardiopulmonary
Inferior myocardial infarction
Congestive heart failure
Pulmonary embolism
Post-tussive

Psychogenic
Anorexia nervosa/bulimia
Chronic vomiting not associated with eating disorders

depending on the location of the obstruction and the time of the last food taken. Feculent vomitus suggests a gastrocolic fistula or a fistula between the colon and duodenum. Motility disorders producing stasis and bacterial overgrowth may also be associated with feculent vomitus.

Basic Science

The resting tone of the gastric fundus establishes a pressure gradient that promotes emptying of liquids into the duodenum. Following ingestion of solids, the pressure at the esophagogastric and gastroduodenal junctions increases and the antral "pump" macerates gastric contents until the mean particle size is less than 1 mm in diameter. Postprandially, the coordinated action of the antrum and pylorus allows small aliquots of gastric contents to be emptied into the duodenum. In the fasting state, the "migrating motor complex" produces regular episodes of increased peristalsis that sweeps undigested solids into the small bowel and propels them distally.

Nausea and vomiting interrupt the intrinsic motor activity of the gastrointestinal tract. With nausea, the normal tone of the fundus and body of the stomach is lost, and pyloric sphincter pressure decreases. Alkaline duodenal contents reflux freely into the distal stomach and may produce vomiting due to local irritation. Retching increases intra-abdominal pressure and promotes duodenogastric and gastroesophageal reflux by simultaneous contraction of the muscles of inspiration, the abdominal wall muscles, and the diaphragm. Vomiting usually occurs at end inspiration when intra-abdominal pressure is highest. The diaphragm abruptly relaxes, and abdominal pressure is suddenly transmitted to the chest. The cardia of the stomach herniates through the diaphragm into the thorax, and stomach contents are projected via the esophagus into the pharynx. Aboral peristalsis has not been demonstrated in humans and is not required to explain the vomiting process.

The physiologic basis for nausea is unknown but may represent conscious awareness of increased activity in the centers controlling the vomiting reflex. The vomiting center (VC) is located in the dorsolateral medulla adjacent to the ascending reticular activating system and the medullary centers controlling cardiovascular and respiratory reflexes. Afferent connections from the cortex, limbic system, hypothalamus, vestibular centers, gut, and other viscera are known. The VC integrates input from central and peripheral afferents and modulates the autonomic and somatic motor response to noxious stimuli. The chemoreceptor trigger zone (CTZ), located in the floor of the fourth ventricle, provides specific receptors for circulating toxins in the blood, and cerebrospinal fluid and relays this information to the VC. Ablation of the CTZ abolishes vomiting produced by increased intracranial pressure or elevated serum levels of digitalis, but does not diminish the emetic response to staphylococcal enterotoxin or gastric distention. In contrast, ablation of the VC destroys the emetic response to vestibular stimulation, exogenous or endogenous toxins and drugs, and mucosal irritation or distention of the gut.

Cells from the area postrema (CTZ) show increased concentrations of catecholamines. Antiemetic medications probably decrease output of the CTZ by antiadrenergic and antidopaminergic effects. This hypothesis could explain why antidopaminergic medications (phenothiazines, butyrophenones, metoclopramide) reduce toxin-induced nausea and vomiting, but are less useful in the treatment of nausea and vomiting produced by obstruction or mucosal irritation. Motion-induced nausea may be decreased by anticholinergic medications (scopalomine) or antihistamines (diphenhydramine), but the mechanism is unknown.

Clinical Significance

Vomiting produces dehydration, metabolic alkalosis, and hypokalemia. One to two liters of stomach secretions containing up to 150 MEq of hydrogen and sodium ion per liter and an isoelectric quantity of chloride ion are produced each day. Distention increases the volume of gastric secretion. During vomiting episodes, bicarbonate ion generated by gastric acid production remains in the vascular space and initiates metabolic alkalosis. Vomiting decreases intravascular volume and increases reabsorption of sodium and water by the proximal renal tubule. Since the intravascular concentration of bicarbonate is increased relative to chloride, high concentrations of bicarbonate are delivered to the kidney. Bicarbonate is reabsorbed as the major anion accompanying sodium throughout the renal tubule, and alkalosis is maintained. Increased tubular concentrations of bicarbonate and increased reabsorption of sodium in the distal tubule promote potassium loss in the urine.

The maintenance of alkalosis and hypokalemia depends on gastric chloride loss in the vomitus. Volume replacement with isotonic sodium chloride and potassium repletion with potassium chloride resolve the metabolic derangements produced by vomiting. Treatment with histamine receptor blocking agents (cimetidine or ranitidine) may decrease secretion of stomach acid and lessen the fluid requirements in patients.

Vomiting, particularly protracted or forceful vomiting, can be associated with tears of the proximal stomach mucosa (Mallory–Weiss tear) and, rarely, with rupture of the esophagus into the left pleural space (Boerhaave's syndrome). Herniation of the cardia through the diaphragm during vomiting may be responsible for these lesions.

Aspiration of stomach contents, particularly by patients with depressed central nervous system function, may produce acid burns of the lungs (Mendelsohn's syndrome) or pneumonia due to aspiration of oropharyngeal contents.

References

Ahmed SS, Gupta RC, Brancato RR. Significance of nausea and vomiting during acute myocardial infarction. Am Heart J 1978;95:671–72.

Barnes JH. The physiology and pharmacology of emesis. Molecular Aspects of Medicine 1984;7:397–508.

Feldman M. Nausea and vomiting. In: Sleisenger MH, Fordtran JS, eds. Gastrointestinal disease. Philadelphia: W. B. Saunders, 1983;160–77.

Gibbs D. Nausea and vomiting. Br Med J 1976;2:1489–92.

*Malagelada J-R, Camilleri M. Unexplained vomiting: a diagnostic challenge. Ann Intern Med 1984;101:211–18.

Stoudemire A, Cotanch P, Laszlo J. Recent advances in the pharmacologic and behavioral management of chemotherapy-induced emesis. Arch Intern Med 1984;144:1029–33.

Swanson DW, Swenson WM, Huizenga KA, et al. Persistant nausea without organic cause. Mayo Clin Proc 1976;51:257–62.